Sleep and Pain in Older Adults: The Role of Negative and Positive Affect

Scott Ravyts
Virginia Commonwealth University

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SLEEP AND PAIN IN OLDER ADULTS: THE ROLE OF NEGATIVE AND POSITIVE AFFECT

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University

by

Scott Ravyts
Master of Science, The University of North Carolina at Chapel Hill, May 2015

Major Director: Joseph M. Dzierzewski, Ph.D.
Assistant Professor
Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
December, 2017
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**Abstract**

SLEEP AND PAIN IN OLDER ADULTS: THE ROLE OF NEGATIVE AND POSITIVE AFFECT

By: Scott Ravyts, M.S.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2017

Director: Joseph M. Dzierzewski, Ph.D.

Assistant Professor of Psychology
Department of Psychology

Poor sleep is known to contribute to increased levels of pain. Preliminary findings suggest that negative and positive affect may mediate this relationship. Given that older adults are prone to both sleep disturbance and pain, the main objectives of the present study were to: 1) examine the relationship between sleep and pain in a non-clinical pain sample of community-dwelling older adults and 2) to examine whether negative and positive affect mediate the relationship between sleep and pain. Baseline measures from 82 older adults participating in the Active Adult Mentoring Project (AAMP) were used for secondary data analysis. A daily sleep diary was used to assess sleep efficiency (SE), total wake time (TWT), total sleep time (TST), and sleep quality (SQ). Affect was measured using the Positive and Negative Affect Schedule (PANAS), while pain was assessed on an 11-point Likert-scale. Findings only partially corroborated past research; SE, SQ, and TWT each predicted pain, while TST did not. In addition, neither positive nor negative affect was found to mediate the relationship between sleep and pain. Methodological and theoretical explanation for the lack of significant mediation are
discussed. Nevertheless, the findings suggest that the assessment and treatment of poor sleep among older adults with pain may be clinically relevant.

*Keywords:* sleep, pain, older adults, negative affect, positive affect
Older adults represent one of the fastest growing segments of the United States population with their number expected to nearly double in the next 30 years (Ortman et al., 2014). As adults age, they are faced with an increasing number of competing health concerns. Disrupted sleep and pain are among the most prevalent and functionally impairing challenges faced in late-life (Colten & Altevogt 2006; Patel, Guralnik, Dansie, & Turk, 2013). Although past research has explored the association between sleep and pain, the literature has focused almost exclusively on clinical pain populations (e.g., osteoarthritis, fibromyalgia) and middle-age adults. Given the high comorbidity between poor sleep and pain in older adults, as well as the adverse consequences associated with each condition, greater attention to the relationship between sleep and pain in late-life is warranted. In addition, while preliminary research has recognized the role of negative affect as a compounding factor in the relationship between sleep and pain, the potentially buffering function of positive affect is still poorly understood. This is significant given that negative and positive affect can serve as a risk or protective factors respectively for many health conditions (Steptoe, Dockray, & Wardle, 2009; Wilson, Bienias, Mendes de Leon, Evans, & Bennett, 2003). Thus, the purpose of the proposed study is to expand previous research by exploring the relationship between sleep and pain in community-dwelling older adults, and to further delineate the potential mediating roles of negative and positive affect in this relationship.

**Sleep**

Sleep is a restorative mental and physical state during which an individual becomes inactive and relatively unresponsive to the environment. Due to its restorative processes, sleep is crucial to health and well-being. For example, sleep increases positive affect (Finan, Quartana, & Smith, 2015), optimizes cognitive performance (Richards et al., 2016), facilitates the
consolidation of new information into memory (Diekelmann & Born, 2010), and promotes social functioning (Beattie, Kyle, Espie, & Biello, 2015). Despite the advantages associated with sleep, sleep disturbance is common, especially for adults in late-life.

**Sleep disturbance in older adults.** Due to its many beneficial properties, sleep has been identified as a marker for healthy aging (Driscoll et al., 2008). Yet, as many as 50% of older adults complain of sleep difficulties (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Sleep disturbance in older adults usually includes one or more of the following complaints: difficulty falling asleep or maintaining sleep, frequent nighttime awakenings, early morning awakenings, decreased total sleep time, decreased sleep efficiency (ratio of time spent sleeping over time in bed), and greater frequency and duration of daytime napping (Vaz Fragoso & Gill, 2007).

Sleep disturbance in late-life results in a range of adverse physical, cognitive and psychological consequences. Disrupted sleep is associated with decreased physical activity and an increase in both functional impairment (Chasens, Sereika, Weaver, & Umlauf, 2007) and risk of falls (Brassington, King, & Bliwise, 2000; Stone et al., 2008). Older adults with disrupted sleep are also more vulnerable to cognitive changes including impaired attention difficulty, decreased working memory, slower reaction times, and lower overall cognitive performance levels (Nebes, Buysse, Halligan, Houck, & Monk, 2009). In addition, impaired sleep contributes to significant changes in mood and affect such as increased risk of depression (Cho et al., 2008), anxiety (Magee & Carmin, 2010), and suicidal ideation (Nadorff, Fiske, Sperry, Petts, & Gregg, 2013). Taken together, the combined physical, cognitive, and psychological effects of sleep disturbance are capable of significantly reducing the quality-of-life of older adults (Schubert et
The breadth of adverse consequences associated with sleep disturbance in late-life has produced interest in better understanding the etiology of disrupted sleep.

Contrary to popular belief among both medical providers and older adults, the aging process in itself is not the cause of disrupted sleep in older adults (Foley, Ancoli-Israel, Britz, & Walsh, 2004; Foley, Monjan, Simonsick, Wallace, & Blazer, 1999). Instead, the causes of sleep disturbance are complex and multifactorial. The 3P model highlights the multifactorial etiology of sleep disturbance by describing how predisposing, precipitating, and perpetuating factors interact to contribute to disrupted sleep (Spielman, Caruso, & Glovinsky, 1987). Predisposing and precipitating factors combine to form a stress-diathesis conceptualization of sleep disturbance. In this model, predisposing factors are events or conditions which increase one’s likelihood of experiencing sleep disturbance (i.e., risk factors), while precipitating factors are events or conditions that trigger the onset of sleep disturbance. Finally, perpetuating factors are behaviors which inadvertently maintain sleep disturbance, thereby enabling the condition to become chronic.

For older adults, predisposing factors include changes in circadian rhythm and sleep architecture including decreased REM sleep and slow wave sleep, as well as increased arousal (Ohayon et al., 2004). Precipitating factors in late-life often involve the onset or treatment of a medical condition or psychiatric condition, such as heart disease, depression, or chronic pain (Foley, Ancoli-Israel, Britz, & Walsh, 2004). Perpetuating factors include a range of poor sleep habits such as more frequent daytime napping (Foley et al., 2007), dysfunctional beliefs and attitudes about sleep (Morin, Stone, Trinkle, Mercer, & Remsberg, 1993), and other unsuccessful attempts to obtain better sleep (i.e., staying in bed for longer periods, trying hard to sleep, etc). The combination of predisposing, precipitating, and perpetuating factors associated with sleep in
late-life can impact other medical comorbidities as adults age. For example, greater daytime napping may lead to less physical activity thereby maintaining and exacerbating one’s chronic pain condition in the long-term.

**Pain**

Pain is an unpleasant sensory and emotional experience that is associated with actual or potential tissue damage [International Association for the Study of Pain (IASP), 1994]. Pain can either be considered acute, subacute, or chronic. Acute pain has a sudden onset and is present for less than three months (King, 2007). Subacute pain is a subset of acute pain defined as pain present for more than six weeks but less than three months (King, 2007). Finally, although no universal definition of chronic pain exists, it has typically been defined as pain that persists beyond normal tissue healing, which is approximately three to six months (IASP, 1994).

Pain is rapidly becoming a significant public health issue due to its increased prevalence. An estimated 30% of Americans report experiencing chronic pain which interferes with their daily functioning (Johannes, Le, Zhou, Johnston, & Dworkin, 2010). While elevated rates of pain exist in the general population, older adults are at a greater risk for pain and are disproportionately affected by the consequences of pain in part because they have diminished physiological and psychological resources needed to manage experiences of pain (Karp, Shega, Morone, & Weiner, 2008).

**Pain in older adults.** Acute pain is a relevant health concern for older adults due to the high frequency of chronic illness and surgical interventions among this population. Moreover, acute pain is a common reason for emergency department visits among older adults (Hwang & Platts-Mills, 2013). Older adults are particularly susceptible to the harmful effects of acute pain due to age-induced physiological changes, such as changes in the function of peripheral and
central nervous system nociceptive pathways (Karp et al., 2008). Despite its prevalence, a lack of literature exists concerning the impact of acute and subacute pain in older adults. Difficulty assessing acute pain in older adults may partially explain the lack of research on this topic (Herr et al., 2004). Nevertheless, the scarcity of research on acute pain in older adults is noteworthy given the high rate of chronic pain in this population, and lack of knowledge regarding how acute pain becomes chronic (Voscopoulos & Lema, 2010).

The prevalence of chronic pain increases significantly with age, with approximately 40% of older adults reporting chronic pain over the past 6 months (Johannes, Le, Zhou, Johnston, & Dworkin, 2010). Chronic pain in late-life manifests in variety of forms; however, the most commonly reported pain complaints include osteoarthritis, back pain, musculoskeletal pain, peripheral neuropathic pain, and chronic joint pain (Denard et al., 2010; Donald & Foy, 2004; Mailis-Gagnon, Nicholson, Yegneswaran, & Zurowski, 2008).

The prevalence of chronic pain in older adults is particularly relevant in light of its association with adverse functional, emotional, and cognitive outcomes. Functional impairments associated with chronic pain are caused by a reduction in ambulation and physical activity (Kop et al., 2005). Although reducing physical activity is considered an appropriate strategy for managing acute pain, in the long-term, this strategy leads to deconditioning, greater gait disturbance, weight gain, and an increased risk for falls (Stubbs et al., 2014).

Chronic pain in older adults also leads to impaired psychological functioning. Depressive and anxiety disorders are both more common in individuals with pain than individuals without pain (Tsang et al., 2008). For example, depression is comorbid with chronic pain in approximately 52% individuals (Bair, Robinson, Katon, & Kroenke, 2003). In addition to being at a higher risk for psychopathology, older adults with pain are more likely to develop
maladaptive beliefs about their condition. This includes the misconception that pain in late-life is inevitable, as well as beliefs that they are unlikely to benefit from treatment (Notcutt & Gibbs, 2010). These erroneous views perpetuate the chronic pain condition by preventing individuals from seeking adequate treatment (Thielke, 2012).

Chronic pain is also associated with poorer cognitive outcomes. For example, chronic pain patients have shown impaired performance on attentional, executive, and general cognitive functioning (Moriarty, McGuire, & Finn, 2011), as well as working memory (Berryman et al., 2013). Taken together, the negative impact of acute, subacute, and chronic pain on numerous aspects of daily functioning has fostered a desire to better understand the causes and contributions to pain in older adults.

Contrary to popular belief among both older adults and medical providers, pain is not an inevitable part of the aging process (Gignac et al., 2006; Thielke, 2012). Instead, it is the direct result of pathology of a physical process (e.g., muscle tissue damage, nerve damage). This pathology can cause nociceptive pain, neuropathic pain, or mixed pain. Nociceptive pain is often described as sharp, aching, or throbbing pain that is caused by activity in the neural pathways in response to tissue damaging stimuli (Nicholson, 2006). In contrast, neuropathic pain is pain that occurs as a direct result of a dysfunction in the nervous system (Nicholson, 2006). Finally, mixed pain is the combination of nociceptive and neuropathic pain.

Although pain is a direct result of physical pathology and psychological processes, aging significantly restricts older adults’ ability to respond to pain due to limited physiological and psychological resources. The diminished capacity to effectively respond to pain in older adults is driven by homeostenosis. Homeostenosis is the progressive reduction in homeostatic reserve that occurs with age in every organ system (Karp et al., 2008). Factors associated with aging such as
cognitive and physical impairments, increased sensitivity to suprathreshold pain stimuli, medical and psychological comorbidities, and social isolation all contribute to pain homeostasis (Karp et al., 2008). Over the long term, pain further decreases physiological reserves and increases the risk of frailty in older individuals (Shega et al., 2012). Given the association between pain and a range of adverse outcomes in older adults, interventions that minimize pain are warranted.

**Sleep and Pain**

Sleep disturbance and pain are both prevalent in older adults and frequently co-occur (Taylor et al., 2007). While it is well-established that pain and sleep are related, research has just begun to unravel the complex association between pain and sleep impairments (Finan, Goodin, & Smith, 2013; Smith & Haythornthwaite, 2004). Some research supports a bidirectional relationship between sleep and pain while other findings indicate a unidirectional relationship where either sleep increases future pain or pain increases future sleep disturbance. A thorough understanding of the relationship between sleep and pain is particularly relevant for older adults given that they are disproportionately affected by the negative consequences of both sleep and pain impairments.

**Pain predicting sleep.** Relatively few studies support a unidirectional relationship where pain increases future sleep disturbance. Nevertheless, support for this relationship has been shown across different study designs. Experimentally induced painful stimuli has been shown to increase sleep disturbance by causing micro-arousals during the night and diminishing the restorative effects of slow-wave sleep (Drewes, Nielsen, Arendt-Nielsen, Birket-Smith, & Hansen, 1997; Lavigne et al., 2000).

Several longitudinal studies also support a unidirectional relationship between pain and sleep. For example, one study examining individuals with rheumatoid arthritis found that pain...
severity at baseline independently predicted sleep disturbance at a 2-year follow-up (Raymond, Nielsen, Lavigne, Manzini, & Choinière, 2001). Similarly, initial pain severity and depression predicted future sleep disturbance in individuals with orofacial pain (Riley et al., 2001).

**Sleep predicting pain.** Several studies examining both clinical and non-clinical pain populations support a unidirectional association between sleep and pain, where disrupted sleep prospectively predicts pain intensity. Experimentally induced partial sleep deprivation in healthy subjects is associated with an increase in spontaneous bodily pain (Haack & Mullington, 2005). Similarly, participants with rheumatoid arthritis with one night of sleep restriction reported significantly higher levels of pain, fatigue, depression, and anxiety (Irwin et al., 2012). Finally, a study that induced sleep deprivation by either decreasing the opportunity for sleep or through forced awakenings throughout the night, found that participants who experienced forced awakenings were more likely to report next-day spontaneous pain compared to both healthy individuals who slept continuously or had a restricted opportunity to sleep (Smith et al., 2007).

Large populations based studies also support a unidirectional relationship between sleep and pain. For example, in a large non-clinical pain sample, baseline insomnia symptoms predicted incident cases of both tension and migraine headaches at an 11-year follow-up (Ødegaard, Sand, Engstrøm, Zwart, & Hagen, 2013). Similarly, headache-free individuals were found to have greater incident cases of headaches at a 1-year follow-up if insomnia symptoms were present at baseline; while the absence of insomnia in individuals with headaches was associated with a greater chance of remission at 1 year (Boardman, Thomas, Millson, & Croft, 2006). Two other large population-based studies found that individuals who reported disrupted sleep at baseline were significantly more likely to be diagnosed with chronic widespread pain at a 15-month follow-up (Gupta et al., 2007) and fibromyalgia at a 10-year follow-up (Mork &
Nilsen, 2012). Finally, another population-based study found that impaired sleep quality at baseline predicted the onset of chronic musculoskeletal pain at a 17-year follow-up (Nitter, Pripp, & Forseth, 2012).

In line with these longitudinal studies, several microlongitudinal studies support a unidirectional relationship between sleep and pain. Sleep disturbance predicts next-day pain in older adults with insomnia (Dzierzewski et al., 2010), as well as adults with depression (Chung & Tso, 2010). Similarly, sleep quality predicts next-day pain in patients with comorbid pain and insomnia (Tang, Goodchild, Sanborn, Howard, & Salkovskis, 2012).

Finally, if disrupted sleep leads to greater pain intensity then increased restorative sleep may be an avenue to reduce or even prevent chronic pain. Davies et al. (2008) found evidence to support this hypothesis by finding that self-reported restorative sleep was independently associated with the resolution of chronic widespread pain. Although further research is warranted, these preliminary findings further support a unidirectional relationship between sleep and pain.

Taken as a whole, these findings suggest that impaired sleep increases the likelihood of pain in pain-free individuals, worsens chronic pain outcomes in individuals with pre-existing conditions, and predicts next-day pain in individuals with pre-existing pain. Finally, preliminary evidence suggests that restorative sleep may facilitate the resolution of chronic pain. Despite the strength of these findings, as well as the variety of study designs supporting these results, other studies indicate that the relationship between sleep and pain may not be unidirectional.

**Bidirectional sleep-pain relationship.** Some evidence supports a bidirectional, or reciprocal relationship between sleep and pain. For example, Alsaadi et al. (2014) found that for individuals with low back pain, a night of poor sleep quality, difficulty falling asleep, waking
after sleep onset, and low sleep efficiency was followed by greater pain intensity during the day. In addition, a day with greater pain intensity was correlated with a decrease in subsequent sleep quality and sleep efficiency, as well as an increase in sleep latency and time awake after sleep onset. Similarly, investigators examining the relationship between sleep and pain in individuals with fibromyalgia found that self-reported disrupted sleep was associated with greater pain the following day and that days with increased pain were associated with greater sleep disturbance the following night (Affleck, Urrows, Tennen, Higgins, & Abeles, 1996).

Despite some support for a bidirectional relationship between sleep and pain, other studies have found more mixed results. Tang et al. (2012) found support for a bidirectional relationship between sleep and pain in adults with insomnia and chronic pain. However, the association was stronger in the direction of poor sleep increasing pain. Another study examining pain in the general population also found a significant reciprocal association between sleep duration and subsequent daily pain (Edwards, Almeida, Klick, Haythornthwaite, & Smith, 2008). Again, the effect was stronger in the sleep to pain direction than the opposite.

Although studies examining the relationship between sleep and pain have resulted in mixed findings, overall, sleep impairments appear to be a stronger and more reliable predictor of pain than pain is of sleep impairments (Finan et al., 2013). This conclusion is supported by micro-longitudinal studies (Chung & Tso, 2010; Dzierzewski et al., 2010), population-based studies indicating that the onset of sleep disturbance reliably predicts pain intensity (Boardman, Thomas, Millson, & Croft, 2006; Gupta et al., 2007; Mork & Nilsen, 2012), and experimental studies suggesting that induced sleep disturbance contributes to the development and maintenance of pain (Haack & Mullington, 2005; Irwin et al., 2012; Smith et al., 2007). Importantly, the recent research designs overcome the methodological limitations associated
with previous studies examining the relationship between sleep and pain but lacked longitudinal and micro-longitudinal data (Smith & Haythornthwaite, 2004). Despite evidence that sleep appears to be a more reliable predictor of pain, further research is warranted to determine whether the relationship between sleep and pain differs according to context and pain severity. In addition, given the high comorbidity of pain and sleep disturbance, as well as the elevated prevalence of each condition with affective disturbances, greater research exploring the connection between sleep, pain, and affect is warranted.

**Affect**

Although no universal definition of affect exists, affect has traditionally been defined as “a neurophysiological state consciously accessible as the simplest raw (non-reflective) feelings evident in moods and emotions” (Russell, 2003). Affect is thus present in both moods and emotions. Emotions are affective states which are brief in nature, caused by a specific event, and often accompanied by a facial or behavioral response (Beedie, Terry, & Lane, 2005). In contrast, moods are affective states that are global in nature, last longer, and are temporally distant from the eliciting stimuli (Beedie, Terry, & Lane, 2005). Some researchers argue that moods and emotions are similar but ultimately conceptually different from affect (Ekkekakis, 2013); however, because these terms have been used inconsistently in the literature, they will be used interchangeably in this thesis.

Affect may be positive or negative. Positive affect is a mental state best characterized by pleasurable engagement with the environment and is typically accompanied by either happiness, joy, excitement, or contentment (Pressman & Cohen, 2005). In contrast, negative affect is considered the extent to which a person experiences subjective distress, unpleasant engagement, and emotional pain (Watson, Clark, & Tellegen, 1988). Although positive and negative affect
generally have negative associations with each other, research suggests that these two constructs may not always represent opposite sides of a bipolar continuum (Russell & Carroll, 1999). Positive and negative affect may instead represent a bivariate distribution with partial overlap. That is, the presence of positive affect does not preclude the presence of negative affect and vice versa. Regardless of the overlap between positive and negative affect, evidence indicates that high positive affect provides a buffering effect against adverse events, while high negative affect is associated with poor outcomes (Pressman & Cohen, 2005).

Positive and negative affect influence health through behavioral, cognitive, and biological pathways (Cohen & Rodriguez, 1995). Affect impacts health behaviorally through the use of health practices, (in)appropriate health care utilization, and (in)adequate care. Affect influences health through cognitive pathways by altering the interpretation of physical stimuli and health decision processes. Finally, affect influences health biologically through the activation or deactivation of the hypothalamic–pituitary–adrenal axis and the sympathetic-adrenal-medullary system (Blackburn-Munro, 2004).

**Negative Affect**

Negative affect has been prospectively associated with a variety of health concerns ranging from hypertension, higher drug use, to cardiovascular disease, and other chronic illnesses (Nabi, Kivimaki, Vogli, Marmot, & Singh-Manoux, 2008). In addition, negative affect worsens the prognosis of individuals with pre-existing conditions (Meyer, von Känel, Saner, Schmid, & Stauber, 2015). Given the consequences associated with negative affect in the general population, the role of negative affect is particularly relevant for older adults who are faced with a decreased number of resources to manage competing health demands and daily stressors.
Although negative affect has been found to decrease in late-life, it still has adverse consequences for older adults (Charles, Reynolds, & Gatz, 2001). For example, negative affect has been found to prospectively predict somatic complaints and acute illnesses in late-life (Leventhal, Hansell, Diefenbach, Leventhal, & Glass, 1996). In addition, higher negative affect has been associated with increased mortality risk (Wilson et al., 2003). In contrast, while high negative affect is associated with poor outcomes, low negative affect appears to be beneficial. Low negative affect combined with positive affect, has been associated with lower levels of symptom distress, fewer depressive symptoms, and higher perceived physical and mental health-related quality-of-life in older adults (Hu & Gruber, 2008).

Positive Affect

Although the role of positive affect has traditionally been overlooked, it has received a considerable amount of recent interest, in part due to its association with positive health outcomes. Specifically, positive affect is associated with decreased morbidity and symptom severity across a range of conditions (Pressman & Cohen, 2005). The benefits of positive affect are largely independent of negative affect (Steptoe et al., 2009), indicating that positive affect may have unique properties which protect against physical and psychological decline.

While negative affect decreases along the lifespan, positive affect tends to remain relatively stable with only a slight reduction in very old age. Some researchers even propose the presence of a ‘positivity effect’ in late-life, where older adults attend to and remember positive information more readily than negative information (Reed & Carstensen, 2012). In addition, in contrast to younger adults, older adults are more likely to have more enduring periods of positive affect (Carstensen, Pasupathi, Mayr, & Nesselroade, 2000). Consequently, positive affective states have the potential to have important health benefits for older adults.
Positive affect is associated with a wide range of protective and adaptive outcomes for older adults. For example, positive affect is significantly associated with a reduction in the incident of strokes in older adults (Ostir, Markides, Peek, & Goodwin, 2001), as well as a decrease in hospital readmission rates following a cardiovascular event (Middleton & Keith, 1996). In addition, high positive affect significantly reduces the risk of both frailty and mortality in late-life (Ostir, Ottenbacher, & Markides, 2004; Zhang & Han, 2016). Finally, older adults with high positive affect have demonstrated better functional recoveries from hip fractures than those with low positive affect or depression (Fredman, Hawkes, Black, Bertrand, & Magaziner, 2006). Given the advantages associated with positive affect, and the consequences of negative affect in late-life, greater understanding of the mechanisms that modify affect are warranted.

**Sleep and Negative Affect**

A well-established finding among studies exploring sleep and affect is that sleep disturbance significantly increases negative affect. One of the earliest study examining the role of sleep on negative affect found that disrupted sleep leads to increases in anxiety, confusion, and total mood disturbance (Dinges et al., 1997). More recent studies have supported these conclusions, finding an association between sleep disturbance and an increase in depressive and anxiety symptoms (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007).

Limited research has explored the relationship between sleep and negative affect in older adults. However, preliminary evidence supports an association in the hypothesized direction. For example, one study found that greater reported time awake and lower sleep quality were associated with more negative affect in older adults (McCrae et al., 2008). Another study found that increased sleep onset latency in older adults was associated with greater anxiety (Leblanc, Desjardins, & Desgagné, 2015).
Several underlying mechanisms have been posited to explain how sleep disturbance contributes to negative affect. One proposed theory hypothesizes that poor sleep contributes to a diminished ability to implement inhibitory control over negative affective states by impairing activity in the prefrontal cortex (Dahl & Lewin, 2002). Another theory postulates that sleep disturbance contributes to greater negative affect by adversely impacting REM sleep which serves as an important modulator of affective brain processes (van der Helm & Walker, 2009). Yet another theory suggests that disrupted sleep leads to negative affect by impairing emotional information processing. According to this model, impaired sleep disrupts individuals’ ability to consolidate emotionally salient and adaptive elements, thereby leading individuals to remember more negative experiences (Payne & Kensinger, 2010). Finally, the cognitive-energy model suggests that sleep disturbance results in greater negative affect due to the depletion of cognitive resources necessary for self-regulation and adaptive actions (Zohar, Tzischinsky, Epstein, & Lavie, 2005).

Sleep and Positive Affect

Although the association between sleep and negative affect has received a considerable amount of consideration, the relationship between sleep and positive affect has received substantially less examination. Moreover, the existing literature examining the association between sleep and positive affect has faced numerous limitations, including selection biases and confounding factors (Ong, Kim, Young, & Steptoe, 2016). Despite these limitations, existing studies suggest that sleep has important implications on positive affect.

Preliminary research indicates that sleep is directly associated with positive affect and may serve to buffer against psychological risk factors. Specifically, positive affect has been associated with better sleep quality (Gray & Watson, 2002), sleep efficiency (Jackowska,
Dockray, Hendrickx, & Steptoe, 2011), and fewer sleep problems (MacDonald & Kormi-Nouri, 2013). Some findings support the presence of a bidirectional relationship between sleep and positive affect, where positive affect influences nightly sleep which in turn impacts next-day affect (Kalmbach, Pillai, Roth, & Drake, 2014).

Sleep not only has the possibility of increasing positive affect, but disrupted sleep has been shown to significantly reduce positive affect (Finan, Quartana, & Smith, 2015). In fact, fragmented sleep was found to be more detrimental to positive affect than either partial sleep loss or delayed bed time (Finan et al., 2015). In addition, disrupted sleep is associated with a reduction in both the intensity and frequency of positive affect (Paterson et al., 2011).

Limited research exists examining positive affect and sleep in older adults. One cross-sectional study found that older adults with fewer sleep problems had significantly higher positive affect (Fredman, Gordon, Heeren, & Stuver, 2014). Specifically, older adults with high positive affect reported significantly better subjective sleep quality. Another study found that nights with more awake time or lower sleep quality significantly reduced positive affect the next day (McCrae et al., 2008). Taken together, these findings support the notion that disrupted sleep is associated with less positive affect in older adults.

**Negative Affect and Pain**

In the past, negative affect was traditionally thought of as a by-product of pain, however, it is now well-established that mood adversely impacts pain by altering pain perception (Gaskin, Greene, Robinson, & Geisser, 1992; Janssen, 2002). According to the gate control theory, negative emotions activate the sympathetic arousal system and open the gate in the spinal cord allowing nociceptive information to be processed by central nervous system (Melzack & Wall, 1967). Over time, negative affect can exacerbate the pain condition and contribute to central
sensitivity, an amplification of neural signaling within the central nervous system that elicits pain hypersensitivity (Adams & Turk, 2015).

Negative affect has an adverse role in the context of both acute and chronic pain. Initially, avoidance of painful activities may be adaptive by preventing injury and promoting recovery in the short-term. However, long-term fear of pain, physical activity, or re-injury inadvertently increases functional disability and leads to greater future negative affect (Crombez, Vlaeyen, Heuts, & Lysens, 1999). In line with this notion, investigators have found that greater negative affect predicted higher levels of pain in the subsequent weeks (Zautra, Johnson, & Davis, 2005). The role of negative affect has also been shown in older adults with acute pain. One study found that negative affect accounted for 27% of the variance in pain for older adults following surgery (Feeney, 2004).

**Positive Affect and Pain**

In contrast to negative affect, positive affect has adaptive benefits in the context of pain. For example, positive affect has been shown to be a source of resilience for individuals with chronic pain, where greater positive affect predicted lower levels of pain in subsequent weeks (Zautra et al., 2005). The ability to regulate positive affect levels by maintaining them at or above one’s normal level is also associated with lower pain intensity (Connelly et al., 2007). Finally, positive emotions may facilitate adaptive adaptations to pain, ultimately resulting in better outcomes (Meulders, Meulders, & Vlaeyen, 2014).

Although there is convincing evidence linking positive affect to decreased pain, the mechanisms through which positive mechanisms decreases pain is less clearly understood. Findings indicate that positive affect decreases pain, not through changes in physiological processes, but rather by attenuating pain perception. For example, positive affect experimentally
induced in the context of acute pain significantly reduces pain sensitivity (de Wied & Verbaten, 2001; Roy, Peretz, & Rainville, 2008). This reduction in pain sensitivity has been found across several affective induction modalities (e.g., humorous film clips, pleasant music) and nociceptive stimuli (e.g., cold pressor, nociception flexion reflex) (Finan & Garland, 2015).

The broaden-and-build theory provides another conceptual framework to explain how positive affect may protect against the adverse consequences associated with chronic pain. The model posits that while negative affect constricts attention and cognition resulting in the automatic activation of bottom-up, habitual, and defensive responses, positive affect counteracts this narrowing process by broadening an individual’s awareness (Fredrickson et al., 2004). Specifically, positive affect broadens one’s awareness through the promotion of exploratory thoughts and behaviors. Over time, this increased awareness facilitates flexible responses to challenges associated with pain and promotes the development of a broadened and durable set of behavioral and cognitive repertoires to deal stressors. This may be particularly relevant for older adults who show a significant reduction in pain coping strategies compared to younger adults (Lachapelle & Hadjistavropoulos, 2005). Preliminary evidence supports the broaden-and-build theory in the context of pain. Positive emotions have been shown to counteract habitual modes of thinking characterized by pain catastrophizing and promote cognitive resilience to pain (Ong, Zautra, & Reid, 2010; Zautra et al., 2005).

**Towards an Integrative Conceptual Model of Sleep, Affect, and Pain in Late-Life**

Sleep disturbance, pain, and affective disturbances are all prevalent and pertinent concerns for adults in late-life. While preliminary evidence suggests that altered dopaminergic function may be common factor among all three experiences (Finan & Smith, 2013), the interaction between sleep, affect, and pain is just beginning to be understood. A number of
longitudinal, microlongitudional, and experimental studies have established the role of sleep in the development and maintenance of pain in both healthy individuals and clinical pain samples (Finan et al., 2013). In addition, preliminary studies indicate that negative affect may be a central mechanism through which sleep disturbance alters pain perception. For example, negative affect significantly mediated the relationship between disrupted sleep and pain among both middle-age and emerging adults with a variety of pain conditions (O’Brien et al., 2010; Valrie, Gil, Redding-Lallinger, & Daeschner, 2008). While positive affect is known to have important benefits on health, research exploring the role of positive affect on the sleep-pain relationship is scarce (Finan et al., 2013).

Disrupted sleep interferes with the natural increase in positive affect typically found in late-life. Instead, sleep disturbance is believed to increase negative affect through a number of mechanisms including impaired REM sleep, decreased inhibitory control, impaired emotional information processing, and the depletion of cognitive resources needed to cope with stressors. Affective experiences, either negative or positive, can influence pain intensity both in the short-term and in the long-term. In the short-term, Melzack and Wall’s (1965) gate-control theory posits that negative affect can increase pain perception, while positive affect can decrease pain perception, by modulating pain signals. Over the long-term, negative affect promotes the use of constricted thinking and behavioral repertoires ultimately limiting one’s resources (e.g., personal support, resilience), making it harder to manage pain. In contrast, the presence of positive affect encourages a broadened repertoire of thoughts and behaviors, which over time leads to the presence of enduring resources which can help better manage pain. Figure 1 displays the proposed integrative model of sleep, affect, and pain.
Figure 1. Integrative model of sleep, affect, and pain in late-life.

Note. Dashed black lines represent short-term mechanisms, while sold black lines represent long-term mechanisms. A direct relationship between sleep and pain is also hypothesized but is not represented in this model.

The Present Study

Older adults are the fastest growing segments of the United States population. Although older adults face a number of health consequences as they age, sleep disturbance and pain are among the most prevalent and functionally impairing (Colten et al., 2006; Patel et al., 2013). While previous research has examined the association between sleep and pain, this association has been primarily limited to chronic pain and younger adult samples. In addition, while past research has begun to explore the role of negative affect in the relationship between sleep and pain, the potentially beneficial role of positive affect has received much less consideration. Thus, the purpose of the proposed study is to: a) examine the relationship between sleep and pain in a non-clinical pain sample of community-dwelling older adults, and b) to elucidate key
mechanisms in this relationship by exploring whether positive and negative affect explain the relationship between sleep and pain. Based on the existing literature, it is hypothesized that a) sleep will predict future pain and that b) negative and positive affect will partially mediate the relationship between sleep and pain, with poorer sleep leading to increased negative affect and greater pain and better sleep leading to greater positive affect and decreased pain.
Methods

Participants

This project included secondary analysis of data from the Active Adult Mentoring Project (AAMP). Specifically, the proposed study examined baseline data obtained over seven days prior to participants being randomly assigned to a study arm. The primary aim of the AAMP study was to examine whether a social-cognitive lifestyle intervention could help promote moderate-intensity exercise in older adults. A description of methods pertinent to the present study are provided here, while the complete methods of the AAMP study are presented elsewhere (Buman et al., 2011; Dzierzewski et al., 2014).

Participants were originally recruited between 2006 and 2008 from a university community in the southeastern United States. Participants were recruited through announcements in a local newspaper, a university older adult participant registry, and flyers in community gathering places. To qualify for the study, participants needed to i) be 50 years old or older, ii) have a self-reported sedentary lifestyle [defined by the Physical Activity Guidelines Advisory Committee (2008) as less than 150 min week of moderate or vigorous physical activity during the past six months], iii) be free of medical conditions that would significantly interfere with the ability to complete unsupervised exercise (e.g., major cardiovascular disease, pulmonary disease, recent cancer treatment), and iv) be free of factors that would significantly interfere with study compliance or assessment (e.g., cognitive impairment, psychosis, hearing or speech impairment).

Procedure

Individuals who were interested in participating in the study first completed a brief screening over the phone in order to ensure that they met eligibility criteria. Participants were
then asked to complete pen and pencil sleep and pain measures daily upon awakening for seven consecutive days. Next, participants retroactively assessed their negative and positive affect during the previous week.

**Measures**

**Sleep.** Participants completed a daily sleep diary upon awakening for seven consecutive days. Daily sleep diaries allowed for a variety of sleep parameters to be assessed. Specifically, this included, i) time to fall asleep (SOL), ii) time spent awake after sleep onset (WASO), iii) time in bed (TIB), iv) sleep quality (SQ), and v) sleep efficiency (SE) [the proportion of time spent sleeping (TST) over time in bed (TIB)]. See Appendix A for the complete sleep diary.

**Pain.** Pain was assessed daily over seven days using a single subjective self-report item: “What is your current level of pain?”. Participants recorded their response to this item on a 10-point Likert scale ranging from 0 (no pain) to 10 (worst pain possible). Although brief, this item is able to capture day-to-day variations in pain and meets criteria recommended in a consensus statement by chronic pain researchers (Dworkin et al., 2008). In addition, even a minimal 1.0 change in pain on this scale is associated with relevant changes in pain (Dworkin et al., 2008). Refer to Appendix A for the pain measure.

**Affect.** Affect was measured using the Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988). The PANAS is composed of two mood scales, one measuring positive affect and another measuring negative affect, each scale consisting of 10 items. Participants were asked to rate the extent to which they experienced a variety of affective states (e.g., irritable, excited, alert) in the last two weeks on a Likert scale from 1 (not at all) to 5 (very). The PANAS has been found to be a valid and reliable measure of both negative and positive affect in non-clinical populations (Crawford & Henry, 2004). See Appendix B for the complete PANAS
measure.

**Data Analyses**

Mediation occurs when a predictor variable affects the criterion variable through an intervening variable. While Baron and Kenny’s (1986) three-step approach to mediation has been among the most historically used approaches to mediation, this technique has several limitations. For example, it is among the lowest in statistical power (Fritz & MacKinnon, 2007), fails to test the indirect effect of the independent variable on the criterion variable, and assumes normality of sampling distributions (Hayes, 2009). In order to overcome these limitations, Preachers and Hayes’ (2008) asymptotic bootstrapping approach was used to examine both the direct effect of sleep on pain, as well as the indirect effect of sleep on pain via both negative and positive affect. Specifically, four different parallel mediation models were used. Given that sleep can be quantified in many forms, each mediation model included one of four different sleep parameters as the predictor variable. These sleep parameters included: SE, SQ, TWT, and TST. Each sleep parameter was used individually as the predictor variable, with both negative affect and positive affect used as parallel mediators. Pain was used as the criterion variable for all four models. Prior to being entered into the model, the sleep parameters and both negative and positive affect were grand-mean centered in order to reduce multicollinearity and facilitate the interpretation of the results.

A post-hoc power analysis was conducted using G*Power (Faul, Erdfelder, Buchner, & Lang, 2009). A mediation analysis with 3 predictors, a sample size of 82 participants, an alpha level of .05, and a medium anticipated effect size consistent with previous research (Schrimpf et al., 2015), resulted in a power level of .80. These results suggest an adequate level of power.
Results

Descriptive Statistics

The final analytic sample included 82 community-dwelling adults in mid- to late-life. The majority of participants were female (82.9%) and White (91.4%) with a mean age of 63.37 (SD = 8.58). Participants’ mean SE was 87.44 % (SD = 8.21) and average TWT of 64.01 (SD = 47.84). Specifically, 40% of the sample reported a sleep efficiency rating below 87.5% which has been identified as the threshold for differentiating individuals with insomnia from healthy controls (Natale et al., 2015). Participants’ average pain levels over seven days were was 1.55 (SD = 1.5) suggesting a mild level of pain. Finally, participants reported greater levels of positive affect ($M = 35.34, SD = 6.80$) than negative affect ($M = 17.24, SD = 5.78$) which is consistent with the norms of non-clinical populations (Watson et al., 1988). Complete demographic information and clinical characteristics of participants’ sleep, pain, and affect are presented in Table 1.

Table 1. Participant Descriptive Statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (Std. Deviation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant Demographics</td>
<td></td>
</tr>
<tr>
<td>Age$^a$</td>
<td>63.37 (8.58)</td>
</tr>
<tr>
<td>Education$^a$</td>
<td>16.15 (2.23)</td>
</tr>
<tr>
<td>Gender (Number of Males)</td>
<td>14</td>
</tr>
<tr>
<td>Race/Ethnicity (Number of</td>
<td>7</td>
</tr>
<tr>
<td>Racial/Ethnic Minorities)</td>
<td></td>
</tr>
<tr>
<td>Sleep Characteristics</td>
<td></td>
</tr>
<tr>
<td>Sleep Efficiency$^c$</td>
<td>87.44 (8.21)</td>
</tr>
<tr>
<td>Total Wake Time$^d$</td>
<td>64.01 (47.84)</td>
</tr>
<tr>
<td>Total Sleep Time$^d$</td>
<td>429.32 (56.02)</td>
</tr>
<tr>
<td>Sleep Quality$^e$</td>
<td>3.57 (1.00)</td>
</tr>
<tr>
<td>Pain Characteristics</td>
<td></td>
</tr>
<tr>
<td>Average Pain Rating$^f$</td>
<td>1.55 (1.50)</td>
</tr>
<tr>
<td>Affective Characteristics</td>
<td></td>
</tr>
<tr>
<td>Negative Affect$^g$</td>
<td>17.24 (5.78)</td>
</tr>
<tr>
<td>Positive Affect$^g$</td>
<td>35.34 (6.80)</td>
</tr>
</tbody>
</table>
Notes:

- units of measurement in years
- depression measured on a scale from 0 to 63
- sleep efficiency measured as a percentage
- sleep variables measured in minutes
- sleep quality measured from 0 to 5
- pain rated on a scale from 0 to 10
- affect rated on a scale from 10 to 50

**Sleep Efficiency, Affect, and Pain**

The total effect of sleep efficiency on pain was significant (Path c, $b = -.05, p = .04$). In contrast, the direct effect of sleep efficiency on pain when controlling for both negative and positive affect did not reach statistical significance (Path $c'$, $b = -.04, p = .05$). Moreover, there was not enough evidence to suggest the presence of an indirect effect of SE on pain via either negative affect (Path $a_1 \times b_1$, $b < -.01$, CI: -.02, .01) or positive affect (Path $a_2 \times b_2$, $b < .01$, CI: -.03, .02). Specifically, although the relationship between SE and negative affect was significant (Path $a_1$, $b = -.20, p = .02$), the relationship between negative affect and pain was not (Path b, $b = .02, p = .57$). Finally, both the relationship between SE and positive affect (Path a, $b = .12, p = .25$), as well as the relationship between positive affect and pain were non-significant (Path b, $b = .02, p = .40$). Figure 2 presents the mediational model for SE, affect, and pain.
Figure 2. Mediational Model of Sleep Efficiency, Affect, and Pain

Note. Standardized regression coefficients and standard errors for the relationship between SE and pain via positive and negative affect. The standardized regression coefficient between SE and pain when controlling for negative and positive affect is in parentheses. The indirect association between SE and pain via negative and positive affect are not represented. *p < .05

**Total Wake Time, Affect, and Pain**

In contrast to SE, both the total effect of TWT on pain (Path c, b < .01, p = .04), as well as the direct effect of TWT on pain were significant (Path c’, b < .01, p < .05). However, both the indirect effect of TWT on pain via negative affect (Path a^1 x b^1, b < .001, CI: -.002, .004), as well as the indirect effect of TWT on pain via positive affect failed to reach statistical significance (Path a^2 x b^2, b < -.001, CI: -.004, .0004). Specifically, while there was a significant relationship between TWT and NA (Path a^1, b = .03, p = .03), there was no significant relationship between TWT and PA (Path a^2, b = -.03, p = .18). Finally, there was no significant relationship between either NA and pain (Path b^1, b = .02, p = .56), nor between PA and pain (Path b^2, b = .02, p = .37). Figure 3 displays the mediational model for TWT, affect, and pain.
Figure 3. Mediational Model of Total Wake Time, Affect, and Pain

Note. Standardized regression coefficients for the relationship between TWT and pain via positive and negative affect. The standardized regression coefficient between TWT and pain when controlling for negative and positive affect is in parentheses. The indirect association between TWT and pain via negative and positive affect are not represented. *p<.05

Total Sleep Time, Affect, and Pain

Neither the total effect of TST on pain (Path c, $b < -.01, p = .38$), nor the direct effect of TST on pain was significant (Path $c'$, $b < -.01, p = .53$). In addition, there was not enough evidence to support the presence of an indirect effect of TST on pain via negative affect (Path $a^1 \times b^1, b < .01, CI: -.003, .001$) or positive affect (Path $a^2 \times b^2, b < .001, CI: -.002, .0004$). Specifically, there was no significant relationship between TST and NA (Path $a^1, b = -.02, p = .09$), or between TST and PA (Path $a^2, b = -.01, p = .47$). In addition, the relationship between NA and pain (Path $b^1, b = .03, p = .36$), as well as PA and pain did not reach statistical significance (Path $b^2, b = .01, p = .57$). Figure 4 presents the full mediational model for TST, affect, and pain.
Figure 4. Mediational Model of Total Sleep Time, Affect, and Pain

Note. Standardized regression coefficients for the relationship between TST and pain via positive and negative affect. The standardized regression coefficient between TST and pain when controlling for negative and positive affect is in parentheses. The indirect association between TST and pain via negative and positive affect are not represented. *p< .05

Sleep Quality, Affect, and Pain

Both the total effect of SQ on pain (Path c, $b = -.67, p = .03$), as well as the direct effect of SQ on pain (Path c’, $b = -.70, p = .03$) were significant. Nevertheless, neither the indirect effect of SQ on pain via negative affect (Path $a^1 \times b^1, b = -.04, CI: -.32, .16$), nor the indirect effect of SQ on pain via positive affect reached statistical significance (Path $a^2 \times b^2, b = .08, CI: -.04, .27$). Specifically, while there was a significant relationship between SQ and NA (Path $a^1, b = -3.25, p = .005$), there was no significant relationship between NA and pain (Path $b^1, b = 01, p = .68$). Finally, there was no significant relationship between SQ and PA (Path $a^2, b = 2.79, p = .05$) and between PA and pain (Path $b^2, b = .03, p = .02$). Figure 5 presents the full mediational model for SQ, affect, and pain.
Figure 5. Mediational Model of Sleep Quality, Affect, and Pain

Note. Standardized regression coefficients for the relationship between SQ and pain via positive and negative affect. The standardized regression coefficient between SQ and pain when controlling for negative and positive affect, is in parentheses. The indirect association between SQ and pain via negative and positive affect are not represented. *p < .05, *** p < .001
Discussion

The purpose of the present study was to examine the relationship between sleep and pain among community-dwelling older adults. Specifically, it was hypothesized that a) sleep would predict future pain and that b) negative and positive affect would each partially mediate the relationship between sleep and pain, with poorer sleep leading to more negative affect and increased pain, and better sleep leading to greater positive affect and decreased pain.

The first hypothesis was partially supported; SE and SQ were each negatively associated with pain, while TWT was positively associated with pain. In contrast, TST showed no association with pain. The second hypothesis was not supported. Neither negative affect nor positive affect mediated the relationship between sleep and pain. Specifically, although SE and SQ showed a negative association with negative affect and TWT showed a positive associated with negative affect, no association between TST and negative affect was found. In addition, there was no evidence for an association between each of the four sleep parameters and positive affect. Finally, there was no evidence for an association between either negative or positive affect and pain for all four models examined in the present study.

Sleep and Pain in Older Adults

The negative association between sleep and pain in the present sample is largely consistent with past research. While there is some evidence that sleep and pain are implicated in a bidirectional relationship, with poor sleep contributing to increased pain, and pain impairing future sleep, a recent meta-analysis indicated that disrupted sleep typically precedes increased pain (Finan et al., 2013). Although these findings are robust, they are limited in their scope, with the majority of research examining the relationship between sleep and pain in adults with diagnosed chronic pain conditions (e.g., osteoarthritis, fibromyalgia) or younger adult
populations (Finan et al., 2013; Smith et al., 2005). Thus, the association between sleep and pain found in the heterogeneous sample of community-dwelling older adults in the present study is noteworthy. Specifically, the results extend past research by suggesting that sleep may be associated with pain for older adults across the pain spectrum. Although little is known about how acute pain becomes chronic (Voscopoulos et al., 2010), some research suggests that restorative sleep may help to resolve pain conditions (Davies et al., 2008). Thus, given that older adults are disproportionately affected by both acute and chronic pain (Karp et al., 2008; Johannes et al., 2010), the current findings suggest that sleep deserves greater attention in the context of pain in late-life.

**Sleep, Pain, and Negative Affect in Older Adults**

The failure of negative affect to mediate the relationship between sleep and pain is largely inconsistent with past research. Several prior studies have shown that negative affect mediates the relationship between sleep and pain for individuals across the life-span (Evans, Djilas, Seidman, Zeltzer, & Tsao, 2017; O’Brien et al., 2011; Valrie et al., 2008). However, these studies have been limited to chronic pain samples. Although no relationship was found between negative affect and pain in the present sample, negative affect has reliably been found to be positively associated with greater levels of pain severity among chronic pain samples (Brown, Robinson, Riley, & Gremillion, 1996; Kratz, Davis, & Zautra, 2007). This discrepancy suggests that the deleterious impact of negative affect on chronic pain may be limited or absent in among individuals with low or acute levels of pain. Some research indicates long-term persistent pain may cause significant physiological changes in the brain responsible for negative mood states which can, in turn, exacerbate chronic pain (Wiech & Tracey, 2009). Older adults’ beliefs
regarding the commonplace occurrence of pain in late-life may also help to manage negative affect in the presence of acute or low levels of pain (Thielke, 2012).

Although negative affect has been identified as a mediator of the sleep-pain pathway, prior studies examining sleep, pain, and negative affect have varied widely in their approach. For example, a study treating sleep as a mediator of the pain-depression pathway found that sleep quality mediates the relationship between pain and depression (Miró, Martínez, Sánchez, Prados, & Medina, 2011). Another study examining chronic pain patients found evidence for their model in which pain mediated the relationship between sleep impairments and depressive symptoms (Hamilton et al., 2012). Finally, Finan, and Smith (2012) proposed the vulnerability model of tonic/phasic mesolimbic dopamine dysregulation to explain the connection between sleep, pain, and affect. Specifically, they hypothesize that abnormalities in the mesolimbic dopamine system may perpetuate the symptoms of insomnia, pain, and negative affect, with persistent exacerbations of these symptoms feeding back to promote greater dopaminergic dysregulation. Taken together, these three findings suggest that while sleep, pain, and negative affect may share a significant amount of variance, their temporal associations remain unclear. Thus, sleep, pain, and negative affect may be temporally connected in an alternative manner than originally proposed. For example, pain may mediate the relationship between sleep and negative affect, or a fourth factor, such as dopamine dysregulation, may better explain the connection between poor sleep, pain, and negative affect.

Finally, the role of negative affect as a mediator of the relationship between sleep and pain may be oversimplified. While the present study found that poorer sleep was associated with greater negative affect, the intensity or frequency of negative affect may not be as relevant as the coping strategies employed to manage one’s negative affect. For example, rumination and
catastrophizing are two common coping strategies believed to exacerbate both insomnia and pain (Smith, Perlis, Carmody, Smith, & Giles, 2001; Turner, Holtzman, & Mancl, 2007). Moreover, compared to adaptive coping strategies, the use of maladaptive coping strategies is associated with greater pain intensity even when presented with the same noxious stimuli (Roditi, Robinson, & Litwins, 2009). Thus, the presence of passive coping styles or maladaptive coping strategies may better explain the mechanism through which sleep disturbance influences pain. These findings suggest that future research may benefit from examining the role of different coping strategies on the relationship between sleep and pain.

**Sleep, Pain, and Positive Affect in Older Adults**

A novel aspect of the present study was the examination of positive affect as a potential mediator of the relationship between sleep and pain in late-life. Research exploring the role of positive affect on the relationship between sleep and chronic pain in children suggests that positive affect may have a mediating effect on the sleep-pain pathway (Evans et al., 2017; Valrie et al., 2008). Contrary to the existing literature examining younger populations, positive affect did not mediate the relationship between sleep and pain in the current sample. Nevertheless, several key findings support the role of positive affect as a potential mediator. First, disrupted sleep is known to strongly attenuate positive affect among adults (Finan et al., 2016). Secondly, positive affect has been shown to reduce pain intensity and improve pain related outcomes (Thong, Tan, & Jensen, 2017).

Several possible factors may explain why positive affect did not mediate the relationship between sleep and pain in the current sample. As with negative affect, positive affect may only influence the relationship between sleep and pain for individuals with chronic pain or diagnosed sleep conditions. Additionally, positive affect may be temporally related to sleep and pain in an
alternative way than proposed. For example, high levels of positive affect has been linked with improved sleep outcomes and might subsequently serve as a buffer against pain (Steptoe, O’Donnell, Marmot, & Wardle, 2008). Finally, prior research has traditionally grouped varying forms of positive affect together (e.g., high arousal, mid-arousal, and low-arousal positive affective states) under the assumption that different forms of positive affect are equally beneficial. However, high arousal positive affective states (e.g., active, alert) might differ in their impact on pain compared to mid-arousal (e.g., cheerful, happy) and low-arousal (e.g., calm, relaxed) states. Although limited to experimentally induced pain, preliminary evidence suggests that only relatively high arousal pleasant emotional states inhibit pain, while low arousal positive emotions do not influence pain sensitivity (Rhudy, Bartley, & Williams, 2010). Thus, the type of positive affect found in the present sample may help to explain the current findings.

**Sleep, Pain, and Affective Arousal in Older Adults**

In order to test whether affective arousal level influenced the sleep – pain relationship in the current sample, exploratory analyses were conducted. Positive affective states were subdivided into high arousal (e.g., excited, enthusiastic) and low arousal states (e.g., proud, interested) and included as separate mediators of the sleep-pain pathway. Refer to Appendix C for the list of high and low arousal affective states.

Participants’ average high arousal positive affect was 13.89 (SD = 2.79), while mean low arousal positive affect was 18.40 (SD = 3.78). High arousal positive affect failed to mediate the sleep – pain pathway when high and low arousal positive affect were entered as potential mediators. That is, no indirect effect was found when either SE (b = .001, CI: -.01, .02), TWT (b < -.001, CI: -.005, .001), TST (b < -.001, CI: -.004, .001) or SQ (b = .04, CI: -.14, .37) was entered as the respective independent variable. Similarly, low arousal positive affect also failed
to mediate the sleep–pain pathway. No mediation occurred when either SE \( (b < -.001, \text{CI:} -.02, .02) \), TWT \( (b < .001, \text{CI:} -.003, .004) \), TST \( (b < .001, \text{CI:} -.001, .003) \) or SQ \( (b = .02, \text{CI:} -.36, .44) \) was entered as the respective independent variable. Appendix D displays the full mediational models for sleep, pain, and high and positive affect arousal level.

In another set of exploratory analyses, negative affective states were also subdivided into high arousal (e.g., scared, hostile) and low arousal states (e.g., guilty, ashamed) and included as separate mediators of the sleep-pain pathway. As with positive affect, there was no evidence to suggest that either high arousal negative affect \( (M = 6.42, SD = 2.47) \) or low arousal negative affect \( (M = 2.82, SD = 1.17) \) mediated the relationship between sleep and pain in the present sample.

**Sleep, Higher Levels of Pain, and Affect in Older Adults**

Given that prior research suggests that both negative affect and positive affect mediate the relationship between sleep and pain in chronic pain samples (Evans et al., 2017; Valrie et al., 2008), exploratory analyses including only older adults with average pain levels above mild \( (M > 2) \) were conducted. Despite higher levels of pain \( (M = 3.40, SD = 1.17) \), analyses did not find evidence to suggest that positive or negative affect acted as mediators of the sleep-pain pathway. That is, there was no indirect effect of either SE \( (b = .02, \text{CI:} -.01, .11) \), TWT \( (b = -.002, \text{CI:} -.01, .001) \), TST \( (b = .001, \text{CI:} -.002, .01) \), or SQ \( (b = .12, \text{CI:} -.16, .94) \) on pain via negative affect. Similarly, there was no evidence for an indirect effect of either SE \( (b < -.001, \text{CI:} -.05, .03) \), TWT \( (b < -.001, \text{CI:} -.01, .005) \), TST \( (b < -.001, \text{CI:} -.001, .002) \), or SQ \( (b = .04, \text{CI:} -.18, .57) \) on pain via positive affect. However, these exploratory analyses were underpowered due to the small sample size of individuals reporting greater than mild levels of pain \( (N = 24) \). Thus, these results are subject to a higher probability of a Type II error.
Limitations

Although this research contributes important insights about the relationship between sleep and pain in community dwelling older adults, it is not without limitations. First, due to the cross-sectional nature of the study, no causal conclusions can be drawn between sleep and pain. While the present study suggests that sleep can predict pain, previous research has shown evidence for a bidirectional relationship between pain and sleep (Finan et al., 2013), thus, pain may also serve as a predictor of sleep in the current sample. Similarly, variations in either sleep or pain, may be the driving force behind the relationship between sleep and pain. That is, variations in sleep may increase pain and variations in pain may increase poor sleep (Dzierzewski et al., 2010; Ravyts et al., 2017). Second, both sleep and pain measures were self-reported and thus subject to recall bias. Third, given the relatively low levels of pain intensity found in the present study, the non-significant mediation of positive and negative affect on the relation between pain and sleep may have been the result of a floor effect. Fourth, the physical activity requirements of the parent study may have limited or excluded individuals with more severe pain from participating. Relatedly, given that the purpose of the parent study was to investigate the effects of physical activity on older adults, the current study did not have information on the presence of diagnosed chronic pain conditions among participants. Therefore, the results of the current study may not generalize to older individuals with either high levels of pain or those with chronic pain conditions.

Implications and Future Directions

Despite these limitations, the present study has several potential implications. First, the association between sleep and pain found in the present study suggests that the assessment and treatment of sleep should be routinely incorporated as a part of clinical care for older adults with
pain. Moreover, the association between sleep and pain in the current sample suggests that interventions targeting sleep which also lead to improvements in pain (Vitiello, Rybarczyk, Von Korff, & Stepanski, 2009; Vitiello et al., 2014), would be beneficial even among older adults with low or acute levels of pain. Secondly, the finding that negative and positive affect did not mediate the relationship between sleep and pain suggests that other mechanisms might be more appropriate to target for older adults with comorbid pain and sleep disturbance. For example, other posited mechanisms include pain catastrophizing, self-efficacy, and depression (Chung & Tso, 2010; Miró et al., 2011; Smith et al., 2001).

Given the cross-sectional nature of the current study, future research would benefit from longitudinally exploring the role of sleep on pain in community-dwelling older adults. In addition, although the current study did not find evidence for a mediating effect of positive or negative affect on the relationship between sleep and pain, preliminary research suggests that the role of affect on these two factors should continue to be explored, particularly among chronic pain samples (O’Brien et al., 2010; Valrie et al., 2008). Specifically, future research would benefit from exploring the role of affect among older adults with higher levels of negative affect, such as those with or at risk for a mental health disorder. Relatedly, future research should continue to consider whether the level of affective arousal plays a role in the sleep-pain pathway. A closer examination of the role of maladaptive cognitive coping styles such as pain catastrophizing or rumination may also be warranted given that it may not be the level of negative affect but the coping strategies employed to manage one’s affect that may influence the relationship between sleep and pain. Finally, the varying methodology with which sleep/pain/affect pathways have been examined suggest that future research would benefit from
clarifying the temporal relationship between all three factors by comparing several competing mediational models including sleep, pain, and affect.

**Conclusions**

Overall, this study extended prior research by highlighting the role of sleep as a predictor of pain in a heterogeneous sample of community-dwelling older adults. In addition, although negative and positive affect did not act as mediators of the sleep-pain pathway in the present sample, several promising avenues of research were outlined to further explore the impact of affect, if any, on sleep and pain in older adults. Given the high degree of overlap between sleep disturbance, pain, and mood dysregulation in late-life, a better understanding of how these three factors relate could have important clinical implications.
References


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Appendix A: Daily Pain and Sleep Diary

DAILY RECORD

(Indicate Date mm/dd/yy) __________________________________________________

Please answer this questionnaire **WHEN YOU AWAKEN IN THE MORNING.** Please enter **yesterday’s day and date above,** and provide the information requested below.

**Instructions.** The next two items ask you to rate how you are feeling. Circle the **one number** that best describes your answer.

1. **Pain level:** On a scale of zero to ten, where zero means no pain and ten equals the worst possible pain, what is your current pain level? (Circle the best answer)

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pain</td>
<td>Mild</td>
<td>Moderate</td>
<td>Severe</td>
<td>Worst possible pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Instructions.** The next questions ask about your sleep yesterday. Please answer the following questions for yesterday. (Indicate yesterday’s day and date? __________________________
e.g., Tues. May 7)

<table>
<thead>
<tr>
<th>SLEEP DIARY</th>
<th>Your answer (yesterday)</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. <strong>NAP</strong> (mins): If you napped yesterday, how long did you nap, in minutes?</td>
<td></td>
<td>1 hr, 12 min</td>
</tr>
<tr>
<td>9. <strong>BEDTIME:</strong> What time did you enter bed for the purpose of sleeping last night?</td>
<td></td>
<td>11:37 pm</td>
</tr>
<tr>
<td>10. <strong>TIME TO FALL ASLEEP</strong> (mins): Counting from the time you wished to fall asleep, how many minutes did it take you to fall asleep?</td>
<td></td>
<td>20 min</td>
</tr>
<tr>
<td>11. <strong>AWAKENINGS:</strong> How many times did you awaken during the night?</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>12. <strong>WAKE TIME</strong> (middle of night): What is the total number of minutes you were awake during the middle of the night once you fell asleep? <em>This does not include the time it took to fall asleep at the beginning of the night, or the time you spent awake in bed before getting out of bed in the morning.</em></td>
<td></td>
<td>30 min</td>
</tr>
<tr>
<td>13. <strong>FINAL WAKE-UP:</strong> What time did you wake up for the last time this morning?</td>
<td></td>
<td>7:13 am</td>
</tr>
<tr>
<td>14. <strong>OUT OF BED:</strong> What time did you actually get out of bed this morning?</td>
<td></td>
<td>7:23 am</td>
</tr>
<tr>
<td>15. <strong>QUALITY RATING:</strong> Pick ONE number to indicate your overall QUALITY RATING or satisfaction with your sleep. [1 = very poor; 2 = poor; 3 = fair; 4 = good; 5 = excellent]</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>16. <strong>BEDTIME MEDICATION</strong> (amount and time): List any sleep medication or alcohol taken at or near bedtime, and give the amount and time taken.</td>
<td></td>
<td>Ambien, 11:00 pm</td>
</tr>
</tbody>
</table>
## Appendix B: PANAS Questionnaire

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. **Indicate to what extent you felt this way over the past week.**

<table>
<thead>
<tr>
<th>Number</th>
<th>Word</th>
<th>Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>____</td>
<td>1. Interested</td>
<td>____ 11. Irritable</td>
</tr>
<tr>
<td>____</td>
<td>2. Distressed</td>
<td>____ 12. Alert</td>
</tr>
<tr>
<td>____</td>
<td>3. Excited</td>
<td>____ 13. Ashamed</td>
</tr>
<tr>
<td>____</td>
<td>5. Strong</td>
<td>____ 15. Nervous</td>
</tr>
<tr>
<td>____</td>
<td>7. Scared</td>
<td>____ 17. Attentive</td>
</tr>
<tr>
<td>____</td>
<td>8. Hostile</td>
<td>____ 18. Jittery</td>
</tr>
<tr>
<td>____</td>
<td>9. Enthusiastic</td>
<td>____ 19. Active</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Scale</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Very Slightly</td>
</tr>
<tr>
<td>2</td>
<td>A Little</td>
</tr>
<tr>
<td>3</td>
<td>Moderately</td>
</tr>
<tr>
<td>4</td>
<td>Quite a Bit</td>
</tr>
<tr>
<td>5</td>
<td>Extremely</td>
</tr>
</tbody>
</table>
Appendix C: High and Low Affective Arousal States

Negative Affect – High Arousal States:

- Scared
- Hostile
- Nervous
- Afraid

Negative Affect – Low Arousal States:

- Guilty
- Ashamed

Positive Affect – High Arousal States:

- Excited
- Enthusiastic
- Alert
- Active

Positive Affect – Low Arousal States:

- Interested
- Proud
- Inspired
- Determined
- Attentive
Appendix D: Mediation Models for Sleep, High and Low Arousal Positive Affect, and Pain

a)

Sleep Efficiency → High Arousal PA

\[
a: b = .03, SE = .04 \\
\]

Low Arousal PA → Pain

\[
c: b = -.05*, SE = .02 \\
(c*: b = -.04, SE = .02) \\
b': b < .01, SE = .08
\]

b)

Total Wake Time → High Arousal PA

\[
a': b < .01, SE < .01 \\
\]

Low Arousal PA → Pain

\[
c: b < .01*, SE < .01 \\
(c*: b < .01*, SE < .01) \\
b': b < .01, SE = .08
\]

c)

Total Sleep Time → High Arousal PA

\[
a: b = -.01, SE = .01 \\
\]

Low Arousal PA → Pain

\[
c: b < -.01, SE < .01 \\
(c*: b < -.01, SE < .01) \\
b': b < .02, SE = .08
\]
Note. Standardized regression coefficients for the relationship between a) sleep efficiency, b) total wake time, c) total sleep time, or d) sleep quality and pain as partially mediated by high and low arousal positive affect. The standardized regression coefficient between each sleep parameter and pain, when controlling for high and low arousal positive affect, is in parentheses. *p < .05.
Appendix E: Mediation Models for Sleep, Affect, and Pain Among Older Adults with Greater than Mild Levels of Pain

a) Sleep Efficiency

\[ a^1: b = -3.1, SE = .13 \]
\[ a^2: b = -0.04, SE = .04 \]
\[ b': b = -0.06, SE = .06 \]
\[ c: b = -.01, SE = .04 \]
\[ (c': b = -.03, SE = .04) \]

Negative Affect

Positive Affect

Pain

b) Total Wake Time

\[ a^1: b = .04, SE = .02 \]
\[ a^2: b = -0.03, SE = .03 \]
\[ b': b = -0.05, SE = .06 \]
\[ c: b = -.001, SE = .01 \]
\[ (c': b = -.001, SE = .01) \]

Negative Affect

Positive Affect

Pain

c) Total Sleep Time

\[ a^1: b = -.01, SE = .02 \]
\[ a^2: b = -.01, SE = .02 \]
\[ b': b = -0.03, SE = .04 \]
\[ c: b = -.01, SE = .004 \]
\[ (c': b = -.01, SE = .004) \]

Negative Affect

Positive Affect

Pain
Note. Standardized regression coefficients for the relationship between a) sleep efficiency, b) total wake time, c) total sleep time, or d) sleep quality and pain as partially mediated by negative and positive affect. The standardized regression coefficient between each sleep parameter and pain, when controlling for negative and positive affect, is in parentheses. *p < .05.